ruses, or other unknown factors. Indeed, electron microscopic examination of lymph nodes from eight patients with CSD has shown the presence of herpes-like agents in all. The association of viruses from this group with lymphoproliferative disorders in humans and subhuman primates has been recognized for many years. In order to provide a safeguard against possible infection with adventitious agents, it has been suggested that all cat-scratch antigen be exposed to a "sterilizing dose" of gamma radiation (cobalt-60, 2.5 mr) before use. This proposal seems well-founded and reasonable. Although radiation of the antigen reduces the intensity of the cutaneous response in about a third of patients, modification of the criteria for a positive reaction should overcome this problem.

At present, therefore, it is suggested that the diagnosis of CSD can be established in most cases on epidemiological and clinical grounds, together with elimination of other infectious causes through appropriate laboratory methods. This must include examination for the presence of bacteria, both aerobic and anaerobic, fungi, mycobacteria and viruses through smear, culture, serological and skin testing. If this is not sufficient, a biopsy specimen of an involved node should be taken for histological examination. Only under unusual circumstances should it be necessary to carry out a skin test to increase confidence in the diagnosis. Until the safety and reliability of the irradiated skin test material have been established, its use also should be limited.

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The Fetal Hydantoin Syndrome

AN ASSOCIATION between maternal ingestion of hydantoins during pregnancy and a specific pattern of malformation in offspring was described by Hill and co-workers in 1974 and further delineated by Hanson and co-workers, who referred to this disorder as the fetal hydantoin syndrome. The most frequently noted features of this condition include the following: mild to moderate growth deficiency usually of prenatal onset; mild mental deficiency; craniofacial defects which include microcephaly, a wide anterior fontanel in the newborn period, ocular hypertelorism, a broad depressed nasal bridge with a short nose, low set abnormally shaped ears and a broad alveolar ridge; limb defects including hypoplasia of the distal phalanges with small nails, finger-like thumbs and altered palmar creases; umbilical and inguinal hernias; short neck with a low hairline, and anomalies of the rib, sternum or spine. Less frequent anomalies include cleft lip or palate, cardiovascular defects, duodenal atresia, anal atresia and genital anomalies.

Hanson and co-workers recently have reported the results of a prospective study of 35 infants whose mothers had been treated with hydantoin anticonvulsants during pregnancy. Eleven percent had the fetal hydantoin syndrome while an additional 31 percent had some features compatible with the prenatal effects of hydantoins, the most frequent of which was developmental or mental deficiency. At present, a safe dose of hydantoin anticonvulsants has not been established below which there is no increased teratogenic risk.

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Dangerous or Ineffective Antidotes

MEDICAL SCIENCE sometimes finds that treatments or procedures that have been used for several years actually are ineffective, perhaps even harmful. Such is the case with three "antidotes" that are widely used and suggested by many toxicology texts, first-aid manuals and product labels: the use of table salt in water as an emetic, using acid solutions to neutralize caustic alkalis and the use of the "universal antidote."

Using table salt in water as an emetic has been suggested for decades. Numerous reports of fatalities caused by this seemingly safe emetic can be found in the literature. The ingestion of a hypertonic saline solution will lead to an elevation in serum sodium concentration, particularly in children. This increase in the effective serum osmolarity will promote a shift of water from the intracellular to extracellular spaces—leading to

cellular dehydration, distention of cerebral vessels, and subarachnoid and subdural hemorrhage.

Another harmful procedure is the use of acid solutions to neutralize ingested alkaline caustics. It is true that a weak acid will neutralize an alkali; however, experimental studies have shown that this reaction results in the production of heat severe enough to produce a thermal burn, further damaging the chemically burned mucosal surfaces. Milk, when used as a diluent, will decrease the caustic potential of the alkali while producing no additional heat.

The combination of activated charcoal, tannic acid and magnesium oxide, known as the "universal antidote," has been shown to be ineffective. The charcoal adsorbs the other ingredients making none of them available for their intended use. It should not be recommended or used.

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The Fetal Alcohol Syndrome

THE FETAL ALCOHOL SYNDROME is a pattern of altered growth, morphogenesis and function seen in the offspring of chronically alcoholic women who continue to drink heavily throughout their pregnancy. First set forth in 1973 by Jones and co-workers, the principal features of this disorder include the following: prenatal and postnatal onset growth deficiency, mental retardation with an average IQ of 63, microcephaly, short palpebral fissures, joint contractures, altered palmar crease patterns, cardiac defects and fine motor dysfunction. Approximately 40 percent of infants born to chronically alcoholic women who continue heavy alcohol consumption during pregnancy have serious problems in development. The incidence of serious developmental defects in the offspring of women who drink lesser amounts of alcohol is not known. However, recent evidence suggests that 11 percent of the offspring of women who drink one ounce of absolute alcohol a day during the first trimester of pregnancy have serious problems in development. Nothing is known about the effect of "social drinking" during the first trimester of pregnancy nor are data available on the effect of "binge drinking."

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Reye Syndrome: Evaluation and Treatment of Intracranial Pressure

INCREASED INTRACRANIAL PRESSURE (ICP) secondary to cerebral edema is the major cause of neurological deterioration in patients with Reye syndrome. Increased intracranial pressure should be evaluated early in the course of treatment before irreversible brain dysfunction has occurred. In the presence of pronounced ICP, a lumbar puncture can be hazardous, risking brain-stem herniation. However, when the possibility of meningitis exists, a spinal tap is indicated. Pretreatment with corticosteroids and mannitol may substantially reduce the risk of herniation in these patients. Computerized axial tomography is extremely useful in showing cerebral edema with visualization of diffuse brain swelling and decreased ventricular size, and can usually show if a subdural hematoma is present.

Although independent evaluations should be done in each patient, routine protocol staging will be useful in guiding treatment and in prognosis. Careful monitoring of fluid given intravenously and of osmotic agents such as mannitol (1.5 to 3 grams per kg of body weight given intravenously), urea (1 to 1.5 grams per kg of body weight given intravenously) or glycerol (1.5 grams per kg of body weight given orally via nasogastric tube), and dexamethasone (1 mg per year of age given intravenously every four hours) can be useful in controlling ICP. However, the single most important aspect of reducing ICP is to insure adequate ventilation. Frequent arterial blood gas determinations should be obtained because respiratory intervention may be necessary. Mild hypothermia also may be useful in reducing ICP.

Severe ICP can be monitored with either an epidural or a direct ventricular catheter attached to a pressure transducer. Control of increased intracranial pressure then can be effected using a combination of mannitol and dexamethasone.